Complete Summary

GUIDELINE TITLE

Practice standards for electrocardiographic monitoring in hospital settings: an American Heart Association scientific statement from the Councils on Cardiovascular Nursing, Clinical Cardiology, and Cardiovascular Disease in the Young.

BIBLIOGRAPHIC SOURCE(S)

Drew BJ, Califf RM, Funk M, Kaufman ES, Krucoff MW, Laks MM, Macfarlane PW, Sommargren C, Swiryn S, Van Hare GF. Practice standards for electrocardiographic monitoring in hospital settings: an American Heart Association Scientific Statement from the Councils on Cardiovascular Nursing, Clinical Cardiology, and Cardiovascular Disease in the Young. Circulation 2004 Oct 26;110(17):2721-46. [147 references] PubMed

GUIDELINE STATUS

This is the current release of the guideline.

COMPLETE SUMMARY CONTENT

SCOPE

DISEASE/CONDITION(S)

Conditions for which electrocardiographic monitoring is appropriate, including arrhythmia, myocardial ischemia, and QT prolongation

GUIDELINE CATEGORY

Diagnosis
Evaluation

CLINICAL SPECIALTY

Cardiology
Critical Care
Emergency Medicine
Internal Medicine
Pediatrics

INTENDED USERS

Advanced Practice Nurses
Hospitals
Nurses
Physicians

GUIDELINE OBJECTIVE(S)

- To provide "best practices" for hospital electrocardiographic (ECG) monitoring
- To make recommendations with regard to indications, timeframes, and strategies to improve the diagnostic accuracy of cardiac arrhythmia, ischemia, and QT-interval monitoring

TARGET POPULATION

Children and adults undergoing hospital cardiac monitoring (refer to "Major Recommendations" for specific populations)

INTERVENTIONS AND PRACTICES CONSIDERED

Real-time electrocardiographic (ECG) monitoring*

*Note: This report does not address the recording of standard "snapshot" 12-lead ECGs in hospital settings or Holter monitoring, which is not performed for prospective clinical decision making.

MAJOR OUTCOMES CONSIDERED

Diagnostic accuracy and prognostic significance of electrocardiographic monitoring in terms of patient outcomes (e.g., death, survival, myocardial infarction)

METHODOLOGY

METHODS USED TO COLLECT/SELECT EVIDENCE

Searches of Electronic Databases

DESCRIPTION OF METHODS USED TO COLLECT/SELECT THE EVIDENCE

Not stated

NUMBER OF SOURCE DOCUMENTS

Not stated

METHODS USED TO ASSESS THE QUALITY AND STRENGTH OF THE EVIDENCE

Expert Consensus

RATING SCHEME FOR THE STRENGTH OF THE EVIDENCE

Not applicable

METHODS USED TO ANALYZE THE EVIDENCE

Review

DESCRIPTION OF THE METHODS USED TO ANALYZE THE EVIDENCE

Not stated

METHODS USED TO FORMULATE THE RECOMMENDATIONS

Expert Consensus

DESCRIPTION OF METHODS USED TO FORMULATE THE RECOMMENDATIONS

Published clinical trials in hospital cardiac monitoring are almost nonexistent. For this reason, it is not possible to develop a formal guideline with levels of evidence supported by published research. Nonetheless, it was deemed appropriate, timely, and valuable by members of the present writing group to provide expert opinions based on clinical experience and related research in the field of electrocardiography (ECG).

RATING SCHEME FOR THE STRENGTH OF THE RECOMMENDATIONS

Not applicable

COST ANALYSIS

A formal cost analysis was not performed and published cost analyses were not reviewed.

METHOD OF GUIDELINE VALIDATION

Peer Review

DESCRIPTION OF METHOD OF GUIDELINE VALIDATION

This statement was approved by the American Heart Association Science Advisory and Coordinating Committee on June 29, 2004 and was endorsed by the International Society of Computerized Electrocardiology and by the American Association of Critical-Care Nurses.

RECOMMENDATIONS

MAJOR RECOMMENDATIONS

The rating system used in this statement was devised by the American College of Cardiology Emergency Cardiac Care Committee and consists of the following categories:

Class I: Cardiac monitoring is indicated in most, if not all, patients in this group.
Class II: Cardiac monitoring may be of benefit in some patients but is not considered essential for all patients.
Class III: Cardiac monitoring is not indicated because a patient's risk of a serious event is so low that monitoring has no therapeutic benefit

Cardiac Arrhythmia Monitoring

Class I

Class I includes all patients at significant risk of an immediate, life-threatening arrhythmia. If a patient is required to leave the monitored unit for diagnostic or therapeutic procedures, then cardiac monitoring should be continued with a portable, battery-operated monitor-defibrillator used by a health care provider who is skilled in electrocardiography (ECG) interpretation and defibrillation. These patients are divided into 16 subcategories.

Patients who Have Been Resuscitated from Cardiac Arrest

The patient resuscitated from outpatient or inpatient cardiac arrest is at high risk for recurrence of that event and should continue to be monitored in an intensive care unit while being evaluated for the cause of the event (e.g., hyperkalemia, acute myocardial ischemia) and while corrective/preventive treatment is being instituted. ECG monitoring should continue until an implantable cardioverter defibrillator (ICD) is implanted, unless the patient had a clearly transient, reversible, preventable, and now-corrected cause of the cardiac arrest. Such transient situations are relatively rare.

Patients in the Early Phase of Acute Coronary Syndromes (ST-Elevation or Non-ST Elevation Myocardial Infarction [MI], Unstable Angina/"Rule-Out" MI)

Much of the published data on ECG monitoring of patients with acute MI were collected during an era when treatment, and therefore the natural history, was different from treatment today. Factors such as early mechanical revascularization, nitrates, aspirin and other antiplatelet and antithrombotic agents, beta-blockers, and angiotensin-converting enzyme inhibitors have revolutionized care and have greatly reduced the incidence and time course of complicating arrhythmias. For example, a patient with acute MI who presents early after onset of symptoms to an institution with an immediate percutaneous coronary intervention protocol may receive a definitive therapy (e.g., a stent to an occluded vessel) and be sent home the next day. At the other end of the spectrum are acute MI patients who do not have such definitively successful reperfusion outcomes or who have a more complicated course because of comorbidities, advanced age, or other factors. Thus, one finds a wide range of recommended ECG monitoring time frames, from 24 hours in the former case to ≥72 hours in the latter case.

It is recommended that monitoring begin as soon as the patient presents to the emergency department (ED) and continue uninterrupted for a minimum of 24 hours for uncomplicated acute MI. Because of the possibility of malignant reperfusion arrhythmias, all patients who receive early reperfusion therapy should undergo uninterrupted ECG monitoring, including during intrahospital transport. Bonnemeier et al reported that in patients with a first MI, those with elevated initial troponin values are more likely than those with normal initial troponins to experience malignant reperfusion arrhythmias after primary percutaneous coronary interventions. In patients with a more complicated course, such as those with ongoing or recurrent ischemia, development of acute heart failure or cardiogenic shock, and arrhythmias requiring an intervention such as temporary pacing, defibrillation, or intravenous antiarrhythmics, monitoring should continue for 24 hours after complications have resolved. Patients with unstable angina or "rule-out" MI should undergo cardiac monitoring until infarction has been ruled out and signs (transient ST-T-wave changes) and symptoms (chest pain or anginal equivalent) of myocardial ischemia have been absent for 24 hours.

Patients with Unstable Coronary Syndromes and Newly Diagnosed High-Risk Coronary Lesions

ECG monitoring is indicated for patients with newly diagnosed critical left main coronary artery disease or its equivalent (e.g., proximal left anterior descending and circumflex disease) who are candidates for urgent revascularization. Monitoring should continue uninterrupted while these patients await intervention.

Adults who Have Undergone Cardiac Surgery

ECG monitoring should be performed after uncomplicated cardiac surgery for a minimum of 48 to 72 hours. For patients at high risk for developing postoperative atrial fibrillation, monitoring should continue until hospital discharge. Risk factors for the development of postoperative atrial fibrillation include advanced age, history of atrial fibrillation, presence of valvular disease, and preoperative beta-blocker withdrawal. Creswell et al reported that the incidence of postoperative atrial fibrillation in a sample of >4,000 patients is 32% after coronary artery bypass surgery, 64% after combined bypass and mitral valve replacement surgery, 49% after combined bypass and aortic valve replacement, and 11% after heart transplantation. The incidence of postoperative atrial fibrillation in minimally invasive coronary bypass procedures is not significantly different than it is with traditional techniques.

The onset of atrial fibrillation typically occurs on the second to fourth postoperative day. Funk and coworkers recently reported that the development of atrial fibrillation after cardiac surgery is not uncommon after hospital discharge.

These investigators found that 14% of 302 patients developed atrial fibrillation in the 2 weeks after hospital discharge and that 69% of these episodes were asymptomatic. A predictor of post-discharge atrial fibrillation was a recorded episode of atrial fibrillation while the patient was hospitalized, which provides a rationale for ECG monitoring throughout the entire hospital stay. Other arrhythmias that occur after cardiac surgery are ventricular tachycardia and fibrillation, atrioventricular (AV) block, and sinus node dysfunction.

A recommendation for the improvement of the diagnostic accuracy of postoperative tachyarrhythmias is to take advantage of atrial epicardial pacemaker leads that often are left in place after surgery. When atrial fibrillation has a ventricular response >150 bpm, the R-R intervals vary less noticeably than they do after the ventricular rate is slowed. Thus, clinicians may fail to note the random R-R irregularity that is characteristic of atrial fibrillation, and the rhythm may be misdiagnosed as paroxysmal supraventricular tachycardia. Likewise, atrial activity may not be obvious on the surface ECG in patients who develop atrial flutter. Furthermore, in a patient with preexisting bundle-branch block, the development of a postoperative supraventricular tachyarrhythmia may be difficult to distinguish from ventricular tachycardia. In all of these situations, an accurate diagnosis can be readily made if an atrial electrogram is recorded. The technique for recording an atrial electrogram is described in the subsequent section on cardiac monitoring lead systems.

Children who Have Undergone Cardiac Surgery

In contrast to adults, children who undergo cardiac surgery, typically to repair congenital cardiac defects, are not particularly at risk for postoperative atrial fibrillation. Arrhythmias that are more commonly observed in the pediatric age group are atrial flutter and junctional ectopic tachycardia. In addition, ventricular tachycardia may occur after procedures that involve ventriculotomy or after coronary reimplantation in the arterial switch procedure for transposition. Recording the atrial electrogram using temporary epicardial pacemaker leads may be especially useful for diagnosing arrhythmias in children after congenital heart surgery. For example, an atrial electrogram is valuable in distinguishing junctional ectopic tachycardia from sinus tachycardia.

Patients who Have Undergone Nonurgent Percutaneous Coronary Intervention with Complications

ECG monitoring is indicated for patients with coronary angioplasty, stenting, or both who experience complications in the catheterization laboratory such as vessel dissection or no reflow or who have less-definitive interventional outcomes. Monitoring should be initiated immediately post-procedure and continue for 24 hours or longer if arrhythmias or ST-segment-deviation events occur.

Patients who Have Undergone Implantation of an Automatic Defibrillator Lead or a Pacemaker Lead and are Considered Dependent

Pacemaker dependency is an unstable or absent spontaneous rhythm with hemodynamic instability in the absence of pacing. Lead dislodgement is a well-known although uncommon early complication after insertion of pacemakers, defibrillators, and (more commonly) biventricular pacemakers. Another less common cause of loss of capture is a sudden increase in pacing threshold. Such threshold increases have been largely eliminated with the widespread use of steroid-eluding leads. Another pacemaker problem that can be identified with ECG monitoring and corrected with noninvasive reprogramming includes the failure to sense (in the atrium or ventricles). ECG monitoring of the patient is recommended for 12 to 24 hours after implantation.

Patients with a Temporary Pacemaker or Transcutaneous Pacing Pads

Temporary transvenous pacemakers are associated with a higher risk of loss of capture than are permanent pacemakers. Temporary transvenous lead wires are stiffer than permanent lead wires to facilitate rapid insertion from remote venous access points. In addition, they lack active and passive fixation mechanisms of permanent leads. This makes lead perforation (through the right ventricular free wall or interventricular septum) or lead dislodgement more likely. In addition, no pacemaker output may occur if lead wires become separated from the external pacemaker generator, batteries become depleted, or oversensing occurs because of large P or T waves or extraneous electrical potentials such as muscle artifact or nearby faulty electrical equipment. Therefore, it is recommended that all patients with temporary pacemakers be monitored until pacing is either no longer necessary and the device is removed or replaced with a permanent device. Transcutaneous pacing is subject to the same concerns as those for other temporary pacemakers. In addition, because the pacing artifact is large, it may obscure or mimic the QRS complex, making it difficult to determine the presence of ventricular capture. In such instances, different ECG monitoring leads should be tried to identify a lead that minimizes the pacemaker artifact and maximizes the QRS complex. If no such lead can be identified, then concomitant monitoring with a non-ECG method is recommended (e.g., arterial pressure, pulse oximetry monitoring, or both).

Patients with AV Block

Monitoring is indicated for patients with Mobitz II block, advanced (2:1 or higher) second-degree AV block, complete heart block, or new-onset bundle-branch block in the setting of acute (especially anterior) MI. Sir Thomas Lewis's "law of the heart" states that natural pacemakers from more distal sites in the conduction system tend to be slower and less reliable. Mobitz II AV block, especially with a wide QRS complex, typically results from disease in the distal (i.e., His-Purkinje) system, and thus if complete block develops, then the escape pacemakers tend to be slow and unreliable. Therefore, patients with Mobitz II AV block require intensive monitoring. Mobitz I (Wenckebach) AV block with a narrow QRS complex is typical of a proximal (i.e., AV nodal) site of block, and thus if complete block develops, then the escape pacemakers are faster and more reliable. Because one cannot always predict the outcome of Mobitz I block, these patients should be monitored unless it has been established that the block is a stable long-term condition.

Second-degree 2:1 AV block or AV block with consecutive blocked P waves is not categorized as Mobitz I or II because it does not allow inference about the proximal versus distal site of block. Because some of these rhythms reflect His-Purkinje system disease, monitoring is recommended. For patients with Mobitz II advanced second-degree AV block, or complete heart block, ECG monitoring should be continued until the block resolves or until a definitive therapy (usually implantation of a permanent pacemaker) is implemented.

Patients with Arrhythmias Complicating Wolff-Parkinson-White (WPW) Syndrome with Rapid Anterograde Conduction over an Accessory Pathway

Sudden cardiac death in Wolff-Parkinson-White syndrome is strongly associated with rapid anterograde conduction over the accessory pathway, typically during atrial fibrillation. Other factors that have been implicated include a family history of Wolf-Parkinson-White, syncope, use of digitalis, and presence of multiple accessory pathways. Therefore, monitoring of patients with arrhythmias exhibiting rapid anterograde conduction over an accessory pathway is recommended until a definitive therapy (usually an ablation procedure) is established.

Patients with Long-QT Syndrome and Associated Ventricular Arrhythmias

Torsades de pointes is a life-threatening, hemodynamically unstable polymorphic ventricular tachycardia that is associated with a prolonged QT interval and is typically triggered by a ventricular premature beat arising out of a pause-dependent increase in U wave amplitude. Prolonged runs may degenerate to ventricular fibrillation. The prolonged QT interval, pause-dependent increases in U wave amplitude, polymorphic ventricular premature beats, or ventricular bigeminy often precede by minutes or even hours polymorphic couplets, triplets, and eventually longer runs. Therefore, strict monitoring of these patients is required. A complete discussion of QT interval monitoring is provided in a later section.

Patients Receiving Intraaortic Balloon Counterpulsation

In addition to the need to monitor all patients who are hemodynamically unstable, patients with a balloon pump may benefit from the recognition of and intervention for arrhythmias that may make tracking by the device difficult and thus decrease its effectiveness. ECG monitoring should be continued until the patient is weaned from the intraaortic balloon pump.

Patients with Acute Heart Failure/Pulmonary Edema

A variety of arrhythmias may contribute to or be the primary cause of acute cardiac decompensation (e.g., the development of atrial fibrillation with an uncontrolled ventricular response). Acute heart failure also is a major risk factor for atrial and ventricular arrhythmias. In addition, some therapies for heart failure, especially intravenous positive inotropic drugs (e.g., milrinone, dobutamine), have significant proarrhythmic properties. Because B-type natriuretic peptide (nesiritide) is an arterial and venous dilator that inhibits sympathetic activity, it may be less arrhythmogenic than positive inotropic agents. Burger et al reported that patients with heart failure who were treated with nesiritide were less likely to experience sustained ventricular tachycardia or cardiac arrest than were patients who were treated with dobutamine. Monitoring is valuable for detecting sinus tachycardia that may signal hypotension during administration of nesiritide. Therefore, continuous monitoring is recommended for all patients until the signs and symptoms of acute heart failure have resolved and cardiac monitoring reveals no hemodynamically significant arrhythmias for at least 24 hours.

Patients with Indications for Intensive Care

ECG monitoring is recommended for patients with major trauma, acute respiratory failure, sepsis, shock, acute pulmonary embolus, major noncardiac surgery (especially in older adult patients with a history of coronary artery disease or coronary risk factors), renal failure with electrolyte abnormalities (e.g., hyperkalemia), drug overdose (especially from known arrhythmogenics, e.g., digitalis, tricyclic antidepressants, phenothiazines, antiarrhythmics), and other illnesses. It is estimated that approximately 1 in 5 patients admitted to intensive care will develop significant arrhythmias, most commonly atrial fibrillation or ventricular tachycardia. Clinically significant arrhythmias have been reported in a variety of surgical populations requiring intensive care, for example, patients undergoing major noncardiothoracic surgery, colorectal surgery, and pulmonary surgery. ECG monitoring should be continued until patients are weaned from mechanical ventilation and are hemodynamically stable.

Patients Undergoing Diagnostic/Therapeutic Procedures Requiring Conscious Sedation or Anesthesia

Numerous procedures requiring conscious sedation are performed in hospital settings (e.g., electrocardioversion). ECG monitoring is indicated for all such procedures and should be continued until patients are awake, alert, and hemodynamically stable.

Patients with Any Other Hemodynamically Unstable Arrhythmia

It is important to point out that arrhythmias that are considered benign in an individual without heart disease may be lethal in a patient with significant heart disease. For example, the development of atrial fibrillation in a patient with critical aortic stenosis or hypertrophic cardiomyopathy may cause immediate hemodynamic deterioration. Therefore, a Class II indication for arrhythmia monitoring may appropriately be a Class I indication for patients with heart disease.

Diagnosis of Arrhythmias in Pediatric Patients

In general, the mechanisms of arrhythmias are the same in children as they are in adults; however, the appearance of the arrhythmias on the ECG may differ because of developmental issues such as heart size, baseline heart rate, sinus and AV node function, and autonomic innervation. For example, the distinction between wide and narrow QRS tachycardia must be altered to take into account a child's age. Although a QRS width of >0.12 second defines wide QRS tachycardia in adults, the upper limit of normal in infants is approximately 0.08 second. This discrepancy means that ventricular tachycardia in an infant with a QRS duration of 0.09 second may be misdiagnosed as supraventricular tachycardia, if adult criteria are used. Similarly, the definition of tachycardia based on rate is also age dependent, with the upper limit of typical being higher in infants (158 bpm) as compared with that in teenagers (120 bpm). These differences present significant issues for the computerized arrhythmia detection algorithms in cardiac monitoring systems, as well as for the clinicians who interpret arrhythmias. Typical age-based ECG standards are shown in Table 1 of the original guideline document.

Class II

ECG monitoring may be beneficial in some patients, but it is not considered essential in all. Cardiac monitoring is helpful in the clinical management of Class II patients, but it is not expected to save lives. Cardiac monitoring often takes place in an intermediate care (telemetry) unit. These patients are divided into 10 subcategories.

Patients with Postacute MI

The decision whether to continue monitoring acute MI patients 24 to 48 hours after admission is controversial. On the one hand, analysis of the Global Use of Strategies to Open Occluded Coronary Arteries (GUSTO-III) study data shows that patients who have late ventricular arrhythmias (>48 hours after hospital admission) have a higher mortality at 1 month and 1 year than do patients who have early arrhythmias. Thus, ECG monitoring past 48 hours would likely help to identify a high-risk group that may benefit from more aggressive therapy and closer post-discharge follow-up. On the other hand, although ventricular arrhythmias after 48 hours post-MI have prognostic significance, they seldom occur. Thus, many patients need to be monitored to identify just 1 of these high-risk patients. Most of the risk for major ventricular arrhythmias in the 15,059 GUSTO-III patients occurred during the first 24 hours, after which the hazard curve was flat. Moreover, 95% of major adverse outcomes (death, stroke, or shock) occurred within the first 24 hours.

Predictors of in-hospital sustained ventricular arrhythmias (ventricular tachycardia and fibrillation) have been reported recently for patients with post-ST-elevation MI and post-non-ST-elevation MI. These predictors include previous hypertension, chronic obstructive pulmonary disease, previous MI, ST-segment changes at presentation, higher Killip class, and lower initial systolic blood pressure. Thus, presently, it seems reasonable to continue to monitor post-MI patients with any of these predictors beyond 48 hours until hospital discharge.

Patients with Chest Pain Syndromes

Patients who present to the ED with chest pain but who do not have diagnostic ECG findings or elevated biomarkers often are admitted to a telemetry unit while repeat troponins and signs and symptoms of myocardial ischemia are monitored. Recently, this practice has been questioned. For example, Snider et al reported that in a total of 414 patients consecutively admitted from the ED to a telemetry unit for suspected acute coronary syndromes, 37% had atypical chest pain and normal ECG findings. Arrhythmias were observed in only 8% of this subgroup and only 4 patients had arrhythmia events that led to an intervention. These investigators concluded that patients with atypical chest pain and a normal ECG in the ED were at low risk of life-threatening arrhythmias and that the use of telemetry monitoring in this group should be reevaluated. Estrada et al assessed the role of telemetry in guiding patient management decisions in 2,240 patients admitted to a telemetry unit. They reported that patients admitted for syncope or chest pain syndromes had lower rates of unexpected intensive care transfer and most were unrelated to arrhythmic conditions. They concluded that telemetry was less valuable for clinical decision making in patients with chest pain syndromes. Estrada et al used the same cohort of 2,240 patients to determine whether the

American College of Cardiology cardiac monitoring guidelines accurately stratified patients according to their risks for developing clinically significant arrhythmias in non-intensive care settings. They concluded that patients with chest pain should be moved from Class I to Class II and patients with arrhythmias should be moved from Class II to Class I.

A major limitation of these investigations is that ST-segment monitoring was not performed in the study's telemetry units. Recently, Pelter et al conducted continuous 12-lead ST-segment monitoring in 237 patients who were treated on a telemetry unit for postacute MI or chest pain syndromes. Thirty-nine patients (17%) had $\geq$1 episode of transient myocardial ischemia (see figure 1 of the original guideline document for 12-lead ST segment monitoring data recorded in a 78-year-old man admitted to a telemetry unit with chest pain syndrome and negative troponins). Serious in-hospital consequences (i.e., death, major arrhythmia, cardiogenic shock, acute pulmonary edema, abrupt reocclusion after percutaneous coronary intervention, MI after telemetry admission, or unplanned transfer to the intensive care unit) occurred in 46% of the group with transient myocardial ischemia as compared with 10% in the group without ischemia ($P<0.001$). Patients with transient myocardial ischemia were 8.5 times more likely than those without ischemia to have in-hospital complications (95% CI, 3.7 to 19.7) after investigators controlled for other predictors of adverse outcome (advanced age, radiographic evidence of heart failure, previous MI). In a companion study, Pelter et al reported that the incidence of transient myocardial ischemia in telemetry units is the same as it was in coronary care units (CCUs) in 1999 and that the vast majority of these ST events are clinically silent (proportion of silent ST events: 71% in the telemetry group, 58% in the CCU group).

A more rational approach to making a decision about which patients presenting to the ED with chest pain should be treated in a hospital unit with ECG monitoring is to use an evidence-based prediction tool. The Goldman risk-assessment tool categorizes patients into a high-, moderate-, low-, or very-low-risk group based on initial ECG and history and physical examination findings. Goldman et al found 5 variables to be valuable in predicting the risk of a major adverse event in a large cohort of >10,000 chest pain patients. These predictors were (1) suspected MI on initial ECG (ST-segment elevation of $\geq$1 mm or pathological Q waves in $\geq$2 leads), (2) suspected ischemia on initial ECG (ST-segment depression of $\geq$1 mm or T wave inversion in $\geq$2 leads), (3) systolic blood pressure <110 mm Hg, (4) rales heard above the bases bilaterally, and (5) history of unstable ischemic heart disease (worsening of previously stable angina, new onset of post-MI angina, angina after a coronary revascularization procedure, or pain that is the same as that associated with a previous MI).

Recently, 2 studies reported using the Goldman risk score to determine which ED patients should receive inpatient monitoring on a telemetry unit. Durairaj et al found that among the 318 patients with chest pain who were classified in the very-low-risk category, 0 suffered a major in-hospital complication. Likewise, Hollander et al found that among 1,029 patients who had a low Goldman risk score and negative initial biomarkers, 0 suffered cardiovascular death or a life-threatening ventricular arrhythmia during hospital telemetry monitoring.

In the absence of a prospective randomized clinical trial to determine whether telemetry-guided management improves patient outcomes, it seems reasonable to recommend inpatient ECG monitoring for patients with any sign of ischemia or infarction on the initial ECG, as well as for patients with $\geq$1 evidence-based risk factor (low systolic blood pressure, pulmonary rales, or exacerbation of ischemic heart disease). ECG monitoring should be continued for 12 to 24 hours until acute MI has been ruled out by negative biomarkers.

Patients who Have Undergone Uncomplicated Nonurgent Percutaneous Coronary Intervention

Monitoring in patients who have undergone uncomplicated, nonurgent percutaneous coronary interventions (i.e., not for acute MI) should begin immediately postintervention, but it need not continue after 6 to 8 hours if patients received a stent. Patients who undergo coronary angioplasty without stenting should be monitored for 12 to 24 hours because of the higher incidence of abrupt closure.

Patients who Are Administered an Antiarrhythmic Drug or Who Require Adjustment of Drugs for Rate Control with Chronic Atrial Tachyarrhythmias

The potential benefits of monitoring include (1) detection of a prolonged QT interval response to the drug, (2) assessment of sinus node function after initiating a drug with negative chronotropic properties, especially when the integrity of the sinus node is uncertain, (3) detection of hemodynamic deterioration after initiating an antiarrhythmic drug with negative inotropic properties, especially in patients with compromised left ventricular function (ejection fraction <40%), and (4) assessment of the efficacy of the drug to control the ventricular rate in chronic atrial fibrillation or flutter, especially with increasing patient activity. It should be pointed out that for patients who are administered certain antiarrhythmic drugs with a known high risk of proarrhythmia, ECG monitoring should be considered a Class I rather than a Class II indication (see "QT Interval and ECG Monitoring for Detection of Proarrhythmia").

Patients who Have Undergone Implantation of a Pacemaker Lead and Are Not Pacemaker Dependent

Patients who are not pacemaker dependent have a spontaneous rhythm in the absence of pacing that does not cause hemodynamic instability. Thus, the goal of monitoring pacemaker function in these patients is not to detect and treat life-threatening bradyarrhythmias but to detect pacemaker failure to capture, pace (no output), or sense appropriately. To confirm that pacing function and programming are appropriate, 12 to 24 hours of postprocedural ECG monitoring is recommended.

Patients who Have Undergone Uncomplicated Ablation of an Arrhythmia

Patients undergoing ablation procedures are typically discharged after a short observation period. AV block is a rare complication of radiofrequency ablation for AV nodal reentrant tachycardia, and it often resolves without permanent pacing. Therefore, it is no longer routine practice to monitor such patients. Patients who may benefit from postprocedural ECG monitoring are those who have experienced prolonged rapid heart rates from an incessant tachycardia because they may develop prolonged QT interval and torsades de pointes after ablation therapy. Likewise, torsades de pointes has been reported in patients with chronic atrial fibrillation who have undergone AV junction ablation with the implantation of a pacemaker. Although pacemaker programming to maintain relatively high paced rates is thought to decrease the incidence of this complication, 12 to 24 hours of ECG monitoring is recommended. In addition, patients with significant organic heart disease who undergo ventricular tachycardia ablation warrant postprocedural monitoring for 12 to 24 hours.

Patients who Have Undergone Routine Coronary Angiography

When vascular closure devices are used to seal the groin puncture, patients often can ambulate and be discharged several hours after uncomplicated diagnostic coronary angiography. ECG monitoring may be indicated immediately after the procedure, however, because vasovagal reactions causing symptomatic bradycardia are not uncommon in this setting.

Patients with Subacute Heart Failure

The role of telemetry monitoring in this patient population is unclear. Opasich et al reported on 711 inpatients with heart failure, 199 of whom underwent telemetry monitoring. The decision to use telemetry was related to known arrhythmia (n=82), electrolyte disturbances (n=20), atrial fibrillation (n=12), symptoms (n=48), intravenous dobutamine (n=13), drug control (n=16), or device control (n=8). The investigators determined that treatment was guided by telemetry in only 33 patients (17%). The physicians' perception was that telemetry monitoring was helpful in 70% of patients, however. One reason for this discrepancy may have been that the investigators considered telemetry important in guiding treatment only if it resulted in a change in treatment. It could be argued that telemetry monitoring may have provided documentation for and reassurance about the efficacy of the treatment plan and that no changes in treatment were warranted. In the absence of randomized clinical trials, it seems reasonable to perform ECG monitoring in the subacute phase of acute heart failure while medications, device therapy, or both are being manipulated.

Patients who Are Being Evaluated for Syncope

Many patients with syncope in whom a careful history is taken do not require hospitalization. Patients with syncope of truly unknown origin should have $\geq$24 hours of inpatient monitoring. The diagnostic yield of ECG monitoring in patients with syncope may be low in the absence of a high amount of suspicion about an arrhythmic cause. Kapoor emphasized that in patients with syncope, heart disease is the major predictor of risk for death or significant arrhythmia. When suspicion arises about an arrhythmic cause for the syncope or in patients who have primary electrophysiologic disorders (e.g., conduction system disease, nonsustained ventricular tachycardia, possible pacemaker malfunction), inpatient monitoring is indicated for 24 to 48 hours, or until an arrhythmic cause has been ruled out by invasive cardiac electrophysiological testing.

Patients with Do-Not-Resuscitate Orders with Arrhythmias that Cause Discomfort

Terminally ill patients experiencing palpitations, shortness of breath, anxiety, or all of these symptoms may require arrhythmia management as part of palliative care provision. The goal of cardiac monitoring in these patients is not to prevent or treat life-threatening arrhythmias, but rather to assist in titrating antiarrhythmic drugs for optimum rate control. ECG monitoring can be discontinued when rate control has been achieved.

Class III

The patients included in this class are postoperative patients who are at low risk for cardiac arrhythmias (e.g., young patients without heart disease who undergo uncomplicated surgical procedures); obstetric patients, unless heart disease is present; patients with permanent, rate-controlled atrial fibrillation; patients undergoing hemodialysis (in general, hemodialysis is performed in outpatient settings [the National Kidney Foundation does not mention the need for ECG monitoring during dialysis; see http://www.kidney.org/professionals/kdoqi/index.cfm]); however, when patients have a Class I or II indication and undergo dialysis in the hospital, ECG monitoring is recommended); and stable patients with chronic ventricular premature beats. Malignant ventricular arrhythmias are unlikely to be triggered by ventricular premature beats in the absence of major modulating factors such as acid-base imbalance, electrolyte abnormality, or myocardial ischemia.

ST-Segment Ischemia Monitoring

Beginning in the mid-1980s, cardiac monitoring companies began adding special ST-segment analysis software to their equipment. Although the current generation of monitors provides for computerized ischemia monitoring, many hospital units still lack this capability. It is also important to point out that in most monitors with computerized ischemia monitoring software, a nurse must activate the software for it to work. Therefore, unlike computerized arrhythmia monitoring that is automatically performed, ST-segment ischemia monitoring must in general be manually enabled. Unfortunately, even in hospital units with computerized ischemia monitoring capability, ST-segment monitoring is widely underused. The results of a recent national random survey of 192 nurse leaders in hospital cardiac units revealed that 46% did not use ST-segment monitoring for the detection of myocardial ischemia in patients admitted with acute coronary syndromes. The primary reason listed for nonuse was "lack of physician support." Other reasons included a high number of false ST alarms and lack of education about how to use the technology and what to do in response to ST alarms.

It is important to point out that no randomized clinical trials have been conducted to determine whether the addition of computerized ST-segment ischemia monitoring improves patient outcomes. Thus, the assignment of the following clinical situations to each of the categories (Class I, II, III) is not based on research but rather on the opinions of the expert writing group. In the absence of such research, it would be inappropriate to state that hospitals without ST-segment monitoring capability are delivering substandard care; however, in the opinion of the expert writing group, when aging cardiac monitors need to be replaced, automated ischemia monitoring capability should be considered, especially for hospitals that provide care for a large number of patients with acute coronary syndromes.

Class I

Patients in the Early Phase of Acute Coronary Syndromes (ST-Elevation or Non-ST-Elevation MI, Unstable Angina/"Rule-Out" MI)

Patients with acute coronary syndromes are the highest-priority candidates for ST-segment monitoring. They should be monitored for a minimum of 24 hours and until they remain event-free for 12 to 24 hours. The potential benefits in patients with acute MI include the ability to (1) assess patency of the culprit artery after thrombolytic therapy; (2) detect abrupt reocclusion after primary angioplasty; (3) detect ongoing ischemia (i.e., failed reperfusion therapy), recurrent ischemia, and infarct extension; and (4) detect transient myocardial ischemia. ST-segment monitoring studies of patients hospitalized with unstable angina show that although 80 to 90% of transient ischemic events are asymptomatic, they are nonetheless significant markers for unfavorable short- and long-term outcomes (see Table 2 of the original guideline document for information on prognostic significance of transient myocardial ischemia with ST-segment monitoring in patients hospitalized for unstable angina).

Patients who Present to the ED with Chest Pain or Anginal Equivalent Symptoms

It is not uncommon for patients with acute ST-elevation MI to have an initial ECG that is non-diagnostic for acute ischemia. Investigators who use continuous monitoring have shown that the ST segment often is dynamic in the early hours of acute MI. This pattern of dynamic ST-segment elevation has been termed "intermittent reperfusion" and is thought to represent cycles of thrombotic occlusion and spontaneous reperfusion in early infarction. Seven studies have reported on the frequency of intermittent reperfusion in acute ST-elevation MI. A meta-analysis of these studies indicates that the frequency is 34 to 40% (95% CI). When ST-segment elevation is dynamic, an initial ECG may not exhibit ST-segment elevation if the patient is in a period of resolving ST segments when the standard 12-lead ECG is recorded. It is important to point out that a standard 12-lead ECG provides only a 10-second period of ECG information. Thus, unless continuous ST-segment monitoring is instituted in the ED, it is likely that some patients who would benefit from early reperfusion therapy will go untreated. Tatum et al reported that 1 to 2% of the 3 million chest pain patients sent home from the ED annually may have been discharged in error, and these "missed MI" patients have a mortality rate almost twice that of the chest pain patients who are admitted to the hospital.

ST-segment monitoring for 8 to 12 hours in combination with testing serum biomarkers of injury may be a cost-effective way to triage patients who present to the ED with chest pain. Because many of these patients do not really suffer from acute coronary syndromes, ST-segment monitoring in the ED may be less costly if it results in fewer "rule-out" MI patients being admitted to a monitored hospital unit.

Patients who Have Undergone Nonurgent Percutaneous Coronary Intervention with Suboptimal Angiographic Results

This group includes patients with coronary angioplasty, stents, or both who experience complications in the catheterization laboratory such as vessel dissection or thrombosis or who have less-definitive interventional outcomes. Monitoring should be initiated immediately postprocedure and continue for ≥24 hours if ST events occur. Abrupt reocclusion is most likely to occur early after the procedure, often before the patient has left the cardiac catheterization laboratory or within the first several hours after transfer to a monitored unit. When multilead ECG monitoring is performed during the intervention, documentation of ST-segment deviation during catheter balloon occlusion improves both sensitivity and specificity of interpretation of ST events postintervention.

Patients with Possible Variant Angina Resulting from Coronary Vasospasm

The potential benefits of ST-segment monitoring include the ability to (1) confirm the diagnosis by observing transient ST-segment elevation, (2) predict the culprit artery and proximity of site of vasospasm (if multilead or 12-lead monitoring is being performed), (3) assess the risk for malignant ventricular arrhythmias during vasospasm, and (4) assess the efficacy of therapy with a calcium-channel blocker. ST monitoring should continue until therapy has been initiated and the patient has been ST event-free for 12 to 24 hours.

Class II

Patients with Postacute MI

ST monitoring should not be discontinued in patients who have experienced recurrent chest pain or anginal symptoms or who have had a second elevation in cardiac enzymes indicating infarct extension until they have experienced a 24-hour-long ST event-free period. If the patient has recurrent symptoms of ischemia after ST monitoring is discontinued, then ST monitoring should be restarted. A potential benefit of ST monitoring in the postacute MI period is to assess a patient's readiness for early mobilization and discharge from the hospital. The absence of ischemic events with increasing physical activity in the hospital provides justification for the efficacy of the antianginal regimen and for early discharge of the patient.

Patients who Have Undergone Nonurgent Uncomplicated Percutaneous Coronary Intervention

Although not mandatory for stable patients, if cardiac monitors are equipped with ST monitoring in the postprocedure unit, ST monitoring should be activated in the immediate postintervention period and continued for 4 to 8 hours. To evaluate the need for postangioplasty cardiac monitoring, Li and coworkers reported on the clinical outcome of consecutive patients who were monitored postintervention. ECG monitoring of 135 patients yielded 23 significant findings (e.g., death, emergency bypass operation, or acute MI). Of the 23 patients with adverse hospital outcomes, 22 had a complicated or an unsuccessful intervention. In the 122 patients with successful coronary angioplasty without angiographic evidence of vessel complications or clinical symptoms at the end of the procedure, no significant arrhythmia or acute MI occurred. These investigators concluded that ECG monitoring is not required after successful, uncomplicated coronary angioplasty. Li's study was conducted in the early 1990s, and the subsequent introduction of stents has made the complication of early abrupt vessel closure even rarer. Thus, cardiac monitoring is not considered mandatory for stable postpercutaneous coronary intervention patients, especially those with only stented vessel(s).

An important potential benefit of ST monitoring in the postintervention period is the ability to evaluate chest pain. In a small cohort of patients, Jeremias et al found that approximately 41% of stent patients and 12% of angioplasty patients experienced postintervention chest pain. Noncardiac chest pain may be caused by stretching the coronary vessel during high-pressure balloon inflations or stent deployment. Benign chest pain, nausea, or other nonspecific symptoms also may result from gastrointestinal causes brought on by fasting or esophageal reflux after eating in a near-supine position. The absence of ST-segment deviation in these situations may provide reassurance that such symptoms are not likely ischemic in nature.

Patients at High Risk for Ischemia After Cardiac or Noncardiac Surgery

The potential benefits of ST monitoring after cardiac surgery are to (1) distinguish incisional from ischemic chest pain, (2) assess graft patency and detect reocclusion, and (3) determine whether postoperative cardiac complications (e.g., arrhythmias, heart failure) have an ischemic basis. It is important to point out that experience with ST monitoring after cardiac surgery is limited. Moreover, few if any clinical studies exist to guide clinicians in distinguishing the gradual diffuse ST-T-wave changes that are frequently observed after pericardiotomy from changes that are indicative of acute myocardial ischemia.

The potential benefit of ST monitoring after noncardiac surgery is to detect perioperative ischemia in older adult patients who are at risk of cardiac complications (e.g., patients with left ventricular hypertrophy, coronary artery or peripheral vascular disease, or cardiac risk factors). The American College of Cardiology/ American Heart Association guideline for perioperative cardiovascular evaluation for noncardiac surgical patients supports intraoperative and postoperative ST-segment monitoring in high-risk situations, which they define as patients with emergent major operations (particularly older adults), aortic and other major vascular surgeries, peripheral vascular surgery, and anticipated prolonged surgical procedures associated with large fluid shifts, blood loss, or both.

Mangano et al reported a high-risk period immediately after surgery when the patient emerges from anesthesia and experiences postoperative pain. Such arousal of the sympathetic nervous system is accompanied by an increased heart rate. Therefore, the mechanism of ischemia in the early postoperative period often results from myocardial oxygen demand that exceeds blood flow capability rather than from a coronary occlusion process.

Any adult who is critically ill (especially older adults) and has a high cardiovascular demand may develop myocardial ischemia and associated cardiac complications. Booker et al reported that, of 76 patients admitted to an intensive care unit for noncardiac reasons (after noncardiac surgery or other major illness), 8 developed transient myocardial ischemia with 12-lead ST-segment monitoring, and of these, 6 also developed elevated serum troponin levels. The 8 patients with transient ischemia experienced a total of 37 ST events (average of 9 events per patient during a 24-hour monitoring period). Only 2 ST events were accompanied by chest pain (95% were clinically silent). Of the 8 patients with transient ischemia, 6 experienced cardiac complications, including non-ST-elevation MI, acute heart failure, and symptomatic arrhythmia, and 1 patient died.

Several studies of ST-segment monitoring in patients being weaned from mechanical ventilation have shown an increased failure to wean as well as an increased risk of cardiac complications in patients with ischemic events as compared with those without ischemic events. Therefore, ST-segment monitoring should be considered intra- and postoperatively, continuing for 24 to 48 hours, in patients in any of these high-risk categories.

Pediatric Patients at Risk of Ischemia or Infarction Resulting from Congenital or Acquired Conditions

The use of ST-segment monitoring in the pediatric population has not been extensively studied or documented; however, ischemic mechanisms have been reported in children. These mechanisms include (1) prenatal exposure to cocaine causing coronary vasospasm in infants, (2) cardiotoxicity during the treatment of severe childhood asthma, (3) intraoperative hypoxia during repair of congenital defects, (4) blunt chest trauma, (5) coronary artery disease from Kawasaki disease, (6) acute myocarditis, and a diverse range of other cardiac conditions.

It may not be feasible to perform ST-segment monitoring in hospitalized children because neonatal and pediatric intensive care units may not be equipped with cardiac monitors that have ST-segment measurement software. In addition, little information can be found about the best lead systems for detecting ischemia in the pediatric population or what ECG criteria should be used. For example, the rapid heart rates that are normally observed in pediatric patients may produce nonspecific ST-T-wave changes. Johnsrude et al studied 96 children with documented MI and reported that ST-segment elevation >2 mm was valuable in making the diagnosis. It remains to be seen whether ST-segment monitoring will have a place in pediatric hospital units.

Class III

Patients with Left Bundle-Branch Block

Patients with left bundle-branch block have ST-T waves that markedly deviate in a positive or negative direction, depending on the ECG lead. The steeply sloping ST segments in these patients cause ST amplitude, which usually is measured at a fixed interval after the J point (e.g., 60 milliseconds), to vary with heart rate. Because ST-segment monitoring software triggers an alarm for a change in ST amplitude, such patients have frequent false ST alarms, and this leads to staff fatigue and disenchantment with the technology. Patients with right bundle-branch block usually can be monitored successfully because the ST-T wave is not so extremely deviated; however, patients with frequent intermittent right bundle-branch block should not be monitored because of false ST alarms whenever the block appears or disappears.

Patients with Ventricular Pacing Rhythm

QRS morphology in right ventricular pacing rhythm is similar to the pattern of left bundle-branch block. Thus, the same rationale for not monitoring patients with left bundle-branch block applies to patients with ventricular pacemakers, especially those with rate-adaptive pacing (variable heart rates). Patients especially prone to false ST alarms are those who fluctuate between spontaneous rhythm (with a more typical ST segment) and pacing rhythm (with a deviated ST segment).

Patients with Other Confounding Arrhythmias That Obscure the ST Segment

Patients with coarse atrial fibrillation or flutter may have fluctuating ST-segment amplitudes because of chaotic atrial activity that is measured in the ST segment. Intermittent accelerated ventricular rhythm also may interfere with ST monitoring. This rhythm is not uncommon in patients with ischemic heart disease, and episodes may last for 30 to 90 seconds, which is long enough to trigger an ST alarm.

Patients who Are Agitated

Patients who are restless and confused are difficult to monitor because of frequent false ST alarms that result from a noisy signal.

QT Interval and ECG Monitoring for Detection of Proarrhythmia

Introduction

The QT interval is an indirect measure of ventricular repolarization. Acute increases in the QT interval can be observed in multiple clinical situations and are associated with an increased risk of syncope and sudden death from torsades de pointes ventricular tachycardia. Clinical situations that may lead to QT prolongation include initiation, increased dosage or overdosage of QT-prolonging drugs, ischemia/infarction, electrolyte disorders, sudden decreases in heart rate, and acute neurologic events.

General Considerations in QT Interval Monitoring

The literature lacks consensus about many aspects of QT interval monitoring. For example, it is unclear how the QT interval measurement should be made, what QT interval threshold should be considered dangerously prolonged, whether corrected QT interval measurements are more efficacious in determining risk for torsades de pointes than uncorrected values, what is the best correction formula to use in clinical practice, and much more. Thus, in the section that follows, the recommendations of the present writing group often are based on expert opinion rather than on proven empirical evidence. More important than QT interval monitoring is continuous ECG monitoring with immediate access to defibrillation because certain conditions pose significant risk of life-threatening arrhythmias and cardiac arrest.

The QT interval should be measured from the beginning of the QRS complex to the end of the T wave. Although the onset of the QRS complex is usually readily apparent, the end of the T wave can be difficult to determine. It can be useful to draw a tangent to the steepest downslope of the T wave and define the intersection of this line with the baseline as the end of the T wave. If the T wave is notched, then the end of the T wave should be considered the end of the entire complex. Discrete U waves, which arise after the T wave has returned to baseline, should not be included in the QT interval. It may be difficult to distinguish a prominent U wave fused with the T wave from a bifid T wave that is characteristic of a congenital long QT syndrome.

Because ventricular repolarization time typically increases with slow heart rates and decreases with fast rates, it is assumed that the QT interval should be corrected for heart rate (QT_C) to assess trends in a given patient over time. However, it is important to point out that the QT_C interval has never been validated as a predictor for torsades de pointes. If a patient has an uncorrected QT interval of 0.44 second before initiation of a potentially proarrhythmic agent and has the same value 8 hours later, then the QT_C at these 2 points may be vastly different if the heart rate is different. In this example, if the predrug heart rate were 60 and the postdrug heart rate were 80, then the QT_C measurement before and after the drug would be 0.44 and 0.52 second, respectively.

A normal QT_C is <0.46 second in women and <0.45 second in men. A QT_C >0.50 second in either sex has been shown to correlate with a higher risk for torsades de pointes. Reported cases of drug-induced torsades de pointes indicate that the vast majority occur in patients with QTC >0.50 second. It is important to point out that this rule has exceptions. For example, amiodarone causes marked prolongation of the QT interval but is not associated with a high risk for proarrhythmia. Another problem in recommending a QT prolongation criterion for clinical practice is that no threshold has been established below which QT prolongation is considered free of proarrhythmic risk.

The most commonly used QT correction formula in clinical practice is the one introduced by Bazett, QT_C = QT interval divided by the square root of the R-R interval measured in seconds. The adequacy of Bazett's formula has been questioned because some evidence exists that the formula overcorrects the QT interval at fast heart rates and undercorrects it at low heart rates. In a recent report on the value of QT_C in predicting coronary heart disease in 14,548 healthy men and women, only minor differences were seen in the risk stratification provided by 3 rate correction methods, with the Bazett correction providing slightly better separation. This finding supports the continued use of the Bazett correction method in clinical practice. If a health care professional is uncertain about how to calculate QT_C, a standard 12-lead ECG can be recorded. Standard ECG algorithms provide both uncorrected and corrected QT intervals. If the computer measurement of the uncorrected QT interval is confirmed by manual measurement, then health care professionals can trust the corrected value of the algorithm.

Because the end of the T wave often is obscure, cardiac monitors do not have algorithms to measure QT intervals and sound an alarm for QT prolongation. Thus, manual measurement by a health care professional is necessary. Lead selection for QT interval monitoring should be made by noting which lead of the patient's standard 12-lead ECG has the most well-defined T wave end. The longest QT interval across 12 leads usually is in a mid-precordial lead (typically V3 or V4), presumably because these leads are in close proximity to the heart and thus have large amplitude T waves. Lead II is a commonly used lead in the research literature for measuring QT intervals, and if the patient has a normal T wave axis, then a prominent positive T wave will be present in this lead. Moreover, when U waves are present, they often are separated from the T wave in lead II so that QT measurement rather than QT_U measurement is possible.

Regardless of the choice of lead for cardiac monitoring in an individual patient, it is important to make QT measurements in the same lead over time. When monitoring a patient for drug-induced prolonged QT, the clinician should document QT_C in the patient's medical record by using a rhythm strip example before the drug is initiated and thereafter at least every 8 hours. In addition, the QT_C should be documented before and after increases in drug dosage.

Risk Factors for Torsades de Pointes

For the subsequent Class I, II, and III categories, QT interval monitoring is a higher priority if the patient has risk factors for torsades de pointes. Risk factors include older age, female sex, heart disease (especially left ventricular hypertrophy, ischemia, or low left ventricular ejection fraction), slow heart rate, electrolyte abnormalities (especially hypokalemia or hypomagnesemia), starvation diet, acquired or genetic metabolic impairment, genetic predisposition to QT prolongation (as detected by baseline QT prolongation or family history of syncope, sudden death, or long QT syndrome), and the concomitant use of other drugs that prolong the QT interval or impair their metabolism. In addition, patients with an increased QT interval are at immediate risk of torsades de pointes if they exhibit QT-related arrhythmias including sudden bradycardia or long pauses (e.g., compensatory pauses after ventricular ectopy), enhanced U waves, T wave alternans, polymorphic ventricular premature beats, couplets, and nonsustained polymorphic ventricular tachycardia (see Figure 2 of the original guideline document for a sample ECG that shows arrhythmias associated with prolonged QT interval that place patient at immediate risk for developing torsades de pointes).

Class I

Patients Administered an Antiarrhythmic Drug Known to Cause Torsades de Pointes

Table 3 in the original guideline document lists potentially proarrhythmic drugs generally accepted by authorities to have a risk of causing prolonged ventricular repolarization and torsades de pointes. The antiarrhythmic agents that are the most likely to cause proarrhythmia include quinidine, procainamide, disopyramide, sotalol, dofetilide, and ibutilide. Amiodarone often causes marked QT interval prolongation; however, it has a low frequency of torsades de pointes. The recommended time frames for ECG QT interval monitoring include 48 to 72 hours for patients initiating or increasing therapy with quinidine, procainamide, disopyramide, sotalol, and dofetilide, and 4 to 5 hours for patients who are being treated with ibutilide. In patients who receive ibutilide for the treatment of atrial fibrillation, the most likely time for torsades de pointes to occur is at the time of conversion to sinus rhythm when a pause occurs. Locati et al analyzed the Holter monitor recordings of 12 patients who developed drug-induced torsades de pointes and found that all episodes were preceded by a short-long-short cycle length sequence. In patients who develop a prolonged QT_C >0.50 second, the offending drug should be discontinued and ECG monitoring should continue until the agent washes out and the QT_C is observed to decrease.

Patients who Overdose from a Potentially Proarrhythmic Agent

ECG monitoring of the QT interval should continue until drug levels have decreased and evidence of marked QT prolongation or associated arrhythmias is no longer found.

Patients with New-Onset Bradyarrhythmias

Patients who develop complete heart block or long sinus pauses with sick sinus syndrome are prone to develop torsades de pointes, including those who have undergone ablation of the AV junction to produce complete heart block to counteract uncontrolled rapid heart rates. Monitoring should continue until the bradyarrhythmia has resolved or definitive treatment (e.g., permanent pacing) has been instituted.

Patients with Severe Hypokalemia or Hypomagnesemia

Patients with severe electrolyte disorders, especially when other risk factors for torsades de pointes are present, should be monitored until the disorder is corrected and no QT-related arrhythmias are present.

Class II

Patients who Require Treatment with Antipsychotics or Other Drugs with Possible Risk of Torsades de Pointes

Drugs with moderate QT prolonging potential are generally initiated in the outpatient setting. In those rare individuals with a history of QT prolongation but in whom the addition of these drugs is judged necessary, in-hospital cardiac monitoring may be recommended. These antipsychotics are listed on the University of Arizona Center for Education and Research on Therapeutics web site (http://torsades.org/medical-pros/drug-lists/drug-lists.htm).

Patients with Acute Neurological Events

Patients with subarachnoid hemorrhage are especially prone to QT prolongation; however, they rarely develop torsades de pointes. Sommargren et al analyzed nearly 90,000 12-lead ECGs from 227 patients with subarachnoid hemorrhage monitored continuously in the neurological intensive care unit. During an average of 114 hours of continuous 12-lead ECG monitoring, a prolonged QT_C was present in 73% of the patients and abnormal U waves were present in 20%; however, only 1 patient developed torsades de pointes. Therefore, patients being monitored in a neurological intensive care unit who have a normal QT_C do not require frequent QT interval measurement. Those with a QTC >0.50 second should be monitored for QT-related arrhythmias and further prolongation of the QT interval.

Class III

Healthy Patients Administered Drugs that Pose Little Risk for Torsades de Pointes

ECG monitoring is unnecessary in patients without baseline QT prolongation or other risk factors for torsades de pointes. The drugs that are unlikely to cause torsades de pointes are listed on the University of Arizona Center for Education and Research on Therapeutics Web site.

See the original guideline document for information on cardiac monitoring lead systems and staffing, training, and methods improving quality of ECG monitoring.

Conclusion

Cardiac monitoring was introduced >40 years ago; hence, a body of clinical knowledge and research guides best practices in hospital settings. Moreover, it is a well-established fact that arrhythmia monitoring with immediately available defibrillation has improved survival and patient outcomes. In contrast, less is known about the efficacy of ST-segment ischemia monitoring or QT interval monitoring. A consensus of experts who manage patients with acute myocardial ischemia and proarrhythmia is not a substitute for carefully conducted randomized clinical trials. Still, important clinical decisions are made every day with cardiac monitoring data. For this reason, the present consensus document represents the best currently available sources to guide clinical practice in hospital settings with respect to ECG monitoring in children and adults.

CLINICAL ALGORITHM(S)

None provided

EVIDENCE SUPPORTING THE RECOMMENDATIONS

TYPE OF EVIDENCE SUPPORTING THE RECOMMENDATIONS

Randomized clinical trials in electrocardiographic monitoring are almost nonexistent; therefore, expert opinions are based upon clinical experience and related research in the field of electrocardiography.

BENEFITS/HARMS OF IMPLEMENTING THE GUIDELINE RECOMMENDATIONS

POTENTIAL BENEFITS

Safe and effective electrocardiographic (ECG) monitoring of patients

POTENTIAL HARMS

Not stated

QUALIFYING STATEMENTS

QUALIFYING STATEMENTS

Randomized clinical trials in electrocardiographic monitoring are almost nonexistent; therefore, expert opinions are based upon clinical experience and related research in the field of electrocardiography.

IMPLEMENTATION OF THE GUIDELINE

DESCRIPTION OF IMPLEMENTATION STRATEGY

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INSTITUTE OF MEDICINE (IOM) NATIONAL HEALTHCARE QUALITY REPORT CATEGORIES

IOM CARE NEED

Getting Better
Living with Illness

IOM DOMAIN

Effectiveness

IDENTIFYING INFORMATION AND AVAILABILITY

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The American Heart Association makes every effort to avoid any actual or potential conflicts of interest that may arise as a result of an outside relationship or a personal, professional, or business interest of a member of the writing panel. Specifically, all members of the writing group are required to complete and submit a Disclosure Questionnaire showing all such relationships that might be perceived as real or potential conflicts of interest.

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Print copies: Available from the American Heart Association, Public Information, 7272 Greenville Ave, Dallas, TX 75231-4596; Phone: 800-242-8721

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